Exhibit II

ADDITIONAL EXPERIMENT

Treatment of HIV-1-infected SCID/hu PBL mice with bpV[pic] prevents CD4+ T lymphocyte depletion.

Human PBMC's were obtained from healthy donors subjected to lymphopheresis for 60 minutes. The yield of PBMC's ranged from 1.8 to 2.6 billion cells per donor. Twenty-five ml fractions of the cell suspensions (obtained by lymphopheresis) were layered on cushions (15 ml) of Lymphocyte Separation Medium (Wisent) in 50 ml tubes, which were then centrifuged at room temperature at 750 x g during 25 minutes. The purified PBMC's were collected and washed 3 times in PBS (500 x g, 7 min, at room temperature and resuspended at 10^8 cells/ml in PBS. Twenty SCID (CB17) mice were each injected intraperitoneally with 0.5 ml of the PBMC suspension. Fifteen mice were infected with clinical isolate 93US151 of HIV-1 (5 $TCID_{50}$), within two hours of reconstitution with PBMC's. Five mice were left uninfected as controls. Infected mice were treated with bpV[pic] (0, 0.9 and 4.6 mg/kg of body weight) everyday, starting on the day of infection. On day 13, all mice were sacrificed and peritoneal washes performed on each mouse. Peritoneal cells were washed twice with PBS and the cell pellet incubated for 30 minutes on ice with a mixture of PerCP-labeled anti-human CD3, FITC-labeled anti-human CD4 and phycoerythrin-labeled anti-human CD8 monoclonal antibodies (Becton-Dickinson) or isotype matched control antibodies. After 2 PBS washes, the cells were fixed with 1% paraformaldehyde in PBS for 30 minutes. Cells were analyzed by flow cytometry using an EPICS Elite ESP cytofluorometer (Coulter). Human T lymphocytes were first identified by the expression of the CD3 marker. The percentage of CD4+ and CD8+ T cells was determined following after acquisition of 10,000 CD3+ human cells.

Results from Figure 9 demonstrate that HIV infection, in the absence of treatment, leads to a predictable decline in the percentage of CD4+ human T cells (from 50 to 18%). The data also show that daily administration of bpV[pic] effectively protects against the CD4+ T cell loss that normally occurs during HIV-1 infection.

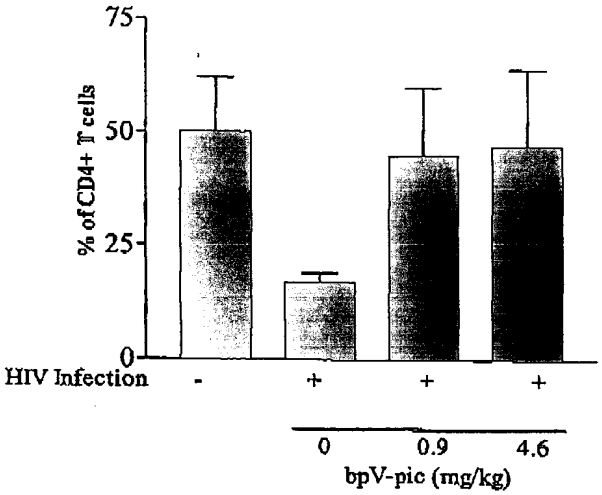

Figure 9